AD-A233 167

DOT/FAA/AM-91/1

Office of Aviation Medicine
Washington, D.C. 20591

The Effect of Simulated Altitude on the Visual Fields of Glaucoma Patients and the Elderly

Van B. Nakagawara

Civil Aeromedical Institute
Federal Aviation Administration
Oklahoma City, Oklahoma 73125

George W. Fulk
Roger W. West

Northeastern State University
College of Optometry
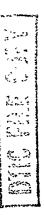Taiequah, Oklahoma 74464-2399

January 1991

Final Report

**DTIC
ELECTE
APRO 5 1991
S B D**

U.S. Department
of Transportation

**Federal Aviation
Administration**

91 4   04 040

NOTICE

This document is disseminated under the sponsorship of
the U.S. Department of Transportation in the interest of
information exchange. The United States Government
assumes no liability for the contents or use thereof.

1. Report No. DOT/FAA/AM-91/1	2. Government Accession No.	3. Recipient's Catalog No.
4. Title and Subtitle THE EFFECT OF SIMULATED ALTITUDE ON THE VISUAL FIELDS OF GLAUCOMA PATIENTS AND THE ELDERLY		5. Report Date January 1991
		6. Performing Organization Code
		8. Performing Organization Report No.
7. Author's) Van B. Nakagawara, O.D.; George W. Fulk, O.D., Ph.D.; Roger W. West, O.D., Ph.D		
9. Performing Organization Name and Address FAA Civil Aeromedical Institute P.O. Box 25082 Oklahoma City, Oklahoma 73125		10. Work Unit No. (TRAIS)
		11. Contract or Grant No.
		13. Type of Report and Period Covered
12. Sponsoring Agency Name and Address Office of Aviation Medicine Federal Aviation Administration 800 Independence Avenue, S.W. Washington, D.C. 20591		
		14. Sponsoring Agency Code

15. Supplementary Notes

This work was performed under task AM-A-89-PHY-144

16. Abstract

This study tests whether mild hypoxia, that is typically encountered in civilian aircraft, causes temporary visual field defects in elderly persons or temporarily increases pre-existing defects in persons with glaucoma. The central 24-2 program on the Humphrey automated perimeter was used to test visual fields in three groups of subjects: six with glaucoma, 12 age-matched controls all of whom were over 44 years of age, and six normal subjects under age 36. Visual fields were tested at ground level and at 10,000 feet in an alternating sequence. A repeated measures design of multiple analysis of variance was used to analyze the data. Altitude was found to have no effect on the visual fields of subjects with glaucoma, age-matched normals, and younger subjects. We found no evidence to suggest a change in the present Federal Aviation Administration standards, which allow a special issuance certificate to persons with glaucoma who wish to obtain medical clearance to operate civilian aircraft. Nor have we found any evidence that should discourage glaucoma patients from flying as passengers.

17. Key Words Vision Glaucoma Visual Fields Certification Hypoxia	18. Distribution Statement Document is available to the public through the National Technical Information Service, Springfield Virginia 22161		
19. Security Classif. (of this report) Unclassified	20. Security Classif. (of this page) Unclassified	21. No. of Pages 22	22. Price

Form DOT F 1700.7 (8-72)   Reproduction of completed page authorized

ACKNOWLEDGEMENTS

THE EFFECT OF SIMULATED ALTITUDE ON THE VISUAL FIELDS OF GLAUCOMA PATIENTS AND THE ELDERLY

INTRODUCTION

A major concern of the Federal Aviation Administration (FAA) Office of Aviation Medicine is the certification of pilot applicants and the determination of appropriate medical standards needed for flight safety. Glaucoma may present a special problem because pilots with glaucoma may give visual field test results that are acceptable at ground level but which may become unacceptable at high altitude due to hypoxia. Another concern is that repeated exposure to high altitudes may accelerate the progression of glaucoma in passengers as well as pilots. If a temporary increase in field loss at typical flight altitudes could be demonstrated, then this would represent a stress on the visual system that, with repeated or prolonged exposures, might cause a permanent increase in visual field loss.

The present study tests whether glaucomatous and/or elderly subjects at an atmospheric pressure simulating the high altitude end of the typical flight environment will suffer temporary scotomas compared with their fields at ground level.

The effect of hypoxia on the nervous system

Due to the high oxygen demands of nervous tissue[1], central nervous system effects are seen even with minimal levels of hypoxia. The degree of these effects depends primarily on the partial pressure of the oxygen (PO_2) breathed and on the length of exposure to that pressure. The directly relevant factor, however, is the partial pressure of oxygen in the arterial blood (PAO_2). PAO_2 is not reduced at a constant rate with increasing altitude. At lower altitudes the reduction is small, but the change in PAO_2 becomes larger above 10,000 feet. For example, PAO_2 at 5,000 feet is about 79% of the sea level value; at 10,000 feet it is about 59% of the sea level value; and at 20,000 feet it is about 33% of the sea level value[2].

McFarland[3] has shown that mental functions start to become impaired at about 10,000 feet. In his studies, short term memory was first seen to decline at 8,000 to 10,000 feet with a more rapid decline after 12,000 feet. Conceptual reasoning began declining at about 12,000 feet with a more rapid fall at 14,000 to 16,000 feet. At about 16,000 feet disorientation, belligerence, euphoria, and a loss of rational judgment may occur.

1

Partial or complete loss of consciousness occurs at about 20,000 feet but this can occur sooner in unacclimatized individuals[1]. Also, exercise has produced loss of consciousness at altitudes as low as 15,000 feet[4]. Carbon monoxide and alcohol, which might be acquired by smoking and drinking, reduce the body's ability to use what oxygen is available and would be expected to produce the above events at lower altitudes[5].

The effect of hypoxia on the visual system

Hypoxia can affect a variety of threshold measures. At about 4,000 feet the absolute dark adapted rod threshold increases until, at 16,000 feet, about twice as much light as at sea level is needed for a threshold response[5]. Ernest and Krill[6] and others have found that the cone as well as rod threshold is increased. Hecht et al.[7] found that lights tend to appear dim, and brightness discrimination becomes obviously reduced at about 8,000 feet. Reduction in brightness discrimination becomes much more marked at 15,000 feet where contrast must be about twice as great as at sea level for threshold. Similar results have been reported by Kohfeld[8]. On the other hand, Kobrick et al.[9] failed to find a reduction in contrast sensitivity even at 25,000 feet.

Visual acuity (VA) can become diminished at about 8,000 feet and at 17,000 feet it approaches only about 50% of what it was at sea level, but these changes occur only in low levels of illumination. With good illumination no VA changes are seen up to 18,000 feet[10].

The visual fields remain grossly normal until about 20,000 feet, after which there is a constriction of the peripheral field, an enlargement of the blind spot, and a development of central scotomas[11]. With more sensitive testing, visual field defects can be found at lower altitudes. Angioscotomas (areas of relative insensitivity around the arterioles of the normal retina) start to enlarge at about 12,000 feet and continue to enlarge until, at about 21,000 feet, the entire visual field is obliterated except for a region 8-10 degrees about the macula[11].

Effects of hypoxia on the eye

Direct retinal damage, mainly in the form of hemorrhages, has occurred in mountain climbers. At about 17,000 feet the disc became hyperemic, the retinal blood vessels dilated and became tortuous, and there was about a 50% incidence of retinal hemorrhages[12]. However, these studies on mountain climbers involved elevations and durations far in excess of what is experienced aboard civilian airplanes. Furthermore, the exercise associated with climbing raised blood pressure and increased oxygen demands. In a study using a hypobaric chamber at 14,500

2

feet for 24 hours, no retinal hemorrhages were seen in spite of a period of maximal exertion[13]. Thus, no retinal hemorrhages would be expected at the low altitude experienced in civilian air travel.

Eysel[14] has determined for the cat that, during anoxia, electrical signals from the various components of the visual system die out over time in the following order: retina (ERG b-wave) first, followed by visual cortex, superior colliculus, lateral geniculate nucleus, and lastly optic tract. Eysel suggested that the extreme sensitivity of the retina to anoxia is due to its greater O_2 consumption/tissue weight for its degree of blood perfusion than other structures such as the cortex.

Much effort has been devoted to determine which retinal layers are most sensitive to hypoxia. This is of particular interest to the present study because glaucoma involves damage to retinal ganglion cells and their axons.

Single-cell recording from cat ganglion cells has shown no changes until the percent oxygen breathed was reduced to under 10%[15,16]. In humans this would correspond to an altitude of 15,000 feet. Alder et al.[15] found that 67% of the ganglion cells showed an <u>increase</u> in spontaneous activity before an eventual decrease (suggesting the loss of inhibition from earlier retinal cells that must be less resistant to hypoxia). On the other hand, Noell[17] reported that a large b-wave can still be seen in man following black-out, indicating that early retinal processes might <u>not</u> be impaired first.

Linsenmeier et al.[18,19] report that, in the cat, tissue PO_2 reaches a minimum at some point in the outer retina. During darkness, this PO_2 gradient across the receptors becomes even steeper due to O_2 consumption by mitochondria deep in the receptor layer, which are most active in the dark. Linsenmeier[19] and Steinberg[20] suggest that the steep PO_2 gradient in the dark makes the receptor outer segments relatively hypoxic and the metabolic rate decreases as a protective measure. This may explain why McFarland et al.[21] and Hecht et al.[7] found that the dark adapted threshold is so sensitive to hypoxia. A very small decrease in PAO_2 causes the already relatively hypoxic rod receptors to decrease their responsiveness to light to conserve O_2.

Clearly, many of the effects of mild hypoxia on the visual nervous system can be explained by receptor dysfunction alone. These effects are measured with great sensitivity by electrophysiological techniques. However, this does not preclude an effect of mild hypoxia on other parts of the visual system. Although it has been difficult to measure these effects in the past, they might be revealed by the highly sensitive visual field testing techniques available today.

The effects of hypoxia on glaucoma

What is the evidence that hypoxia may have a significant effect on glaucomatous visual fields? High altitude might be expected to exacerbate glaucoma in <u>at</u> <u>least</u> two ways--by changing intraocular pressure (IOP), or by reducing PO_2 in blood delivered to the optic nerve.

The literature on the effect of atmospheric pressure on IOP is inconsistent. Mercier et al.[22] reported that in a low pressure chamber simulating 10,000 feet, normal subjects as well as treated and non-treated glaucoma subjects showed a <u>decrease</u> in IOP of about 3-5 mm Hg which continued even into descent. However, Mercier et al.[22] also reported that Furaya and Bucalossi found <u>increases</u> in IOP on ascent.

Payne[23] gives arguments but no data that IOP would not be expected to change with altitude because the eye is an enclosed globe of fluid. On the other hand, when gas bubbles are in the eye (for example after injections of a slow dissolving gas such as perfluoropropane for retinal detachment surgery), ascent by airplane[24] or by car in the mountains[25] can greatly increase IOP.

Epidemiologic data are also available. Payne[23] reported that W.A. Arzabe found no difference in the prevalence of glaucoma in Bolivia (about 3%) for individuals living at about 10,000 feet compared to those living near sea level. Mercier et al.[22] examined 1048 eyes of civilian and military flying personnel and found glaucoma in 2.7%, although their main criterion for glaucoma was a pressure over 21 mm Hg. Fighter, bomber, and test pilots had more glaucoma (3.8%) than other civilian and military categories (1.6%). This finding suggested to these authors the possibiliy that certain flight conditions might increase IOP or otherwise aggravate glaucoma, but they did not elaborate on this.

Whether or not hypoxia is a likely candidate for accelerating the progression of glaucoma or temporarily increasing field loss may depend on whether naturally occurring hypoxia has any role in the etiology of open angle glaucoma. An hypoxic etiology for glaucoma is still maintained as possible by some, but they are in the minority. Anderson[26] suggests that individuals who are abnormally susceptible to optic nerve damage from high IOP may have faulty autoregulation of the blood supply to the optic nerve head.

The primary study that supports the hypothesis that hypoxia can exacerbate field loss typical of glaucoma may be Evans and McFarland[11], in which they note that the loss of visual field caused by the enlargement of angioscotomas during hypoxia follows very closely the pattern of field loss due to glaucoma. Since that study, visual field testing devices have become much more

sophisticated and it might be expected that other subtle field changes may be found with the relatively minor hypoxia associated with typical flight conditions.

It is of interest to test whether eyes with glaucoma-induced visual field defects show significant transient growth in scotomas at elevations commonly encountered by pilots and passengers (a maximum of about 8,000 feet within a pressurized cabin). If this conjecture is supported, it may also be possible that prolonged exposure to the mildly hypoxic conditions that occur in long or repeated flights may cause a more rapid progression of glaucomatous field defects. In addition, what may have been measured as small, relatively safe scotomas at ground level may enlarge and become unsafe in flight.

Hypoxia and age

The age structure of the population of licensed pilots has changed considerably in the last 20 years. The percentage of airmen over 50 years old has more than doubled from 9.5% in 1966 to 21.7% in 1986[27]. Retinal sensitivity, as determined by automated perimetry, declines with age, especially in the periphery[28]. The optic nerve is also known to undergo a reduction in the number of nerve fibers with advancing age[29]. For these reasons we also explored the possibility that age would alter the effect of altitude-induced hypoxia on visual field testing.

METHODS

This study looked for potential effects of altitude on visual fields across two variables between subjects: glaucoma and age. We did this by making two comparisons. First we compared the visual fields of glaucoma subjects and age-matched controls with respect to the possible effects of altitude. Secondly we made a similar comparison between normal older subjects and normal younger subjects. The data for the control group in the first comparison were the same as those used for the older group in the second comparison.

Subjects

For each of the six glaucoma subjects, we selected two age-matched subjects without glaucoma (controls). All subjects in that portion of the study were over 44 years of age (mean 58.4 years). We also tested six subjects without glaucoma under age 36 (mean 30.8 years). Comparison of the results from younger

5

subjects with those of the older control subjects constituted our study of age-dependent effects of altitude on visual fields.

We solicited paid volunteers to participate in the study through private optometry and ophthalmology practices and by placing ads in the local newspapers. To be selected as a subject, an applicant had to fulfill all of the following conditions:

1. pass a medical examination equivalent to a FAA class-three physical which is required to obtain an airman medical certificate (with supplemental EKG, spirometry, urinalysis, and a hearing test);
2. have corrected visual acuity of 20/30 or better in at least one eye;
3. be free of any non-glaucomatous retinal lesions that would be likely to cause a defect in the central 30 degrees of the visual field;
4. be able to respond reliably to the visual field testing, i.e. the false positive and false negative rates needed to be less than 33% and fixation losses less than 20%;
5. be able to be clearly classified as either having glaucoma or being free of glaucoma as defined below.

Glaucoma subjects met all of the following points while control subjects met none of them:

a) a visual field defect typical of glaucoma, i.e. a nasal step and/or a paracentral scotoma;
b) elevated intraocular pressure (IOP) or a history of elevated IOP;
c) evidence of erosion of the rim tissue of the optic nerve apparent when viewed stereoscopically with a slit lamp;
d) currently under treatment for glaucoma.

Procedure

Visual fields were tested with the 24-2 threshold program on the model 630 Humphrey Field Analyzer. This program estimated the retinal threshold at 51 points within the central 24 degrees (including the fovea, excluding the blind spot), and two additional points on either side of the horizontal meridian at 28 degrees on the nasal side for a total of 53 points. Thresholds that deviated by more than five decibels (dB) from the expected value were estimated a second time. We used the average threshold value for all points thresholded twice. The testing sequence took about 10 minutes per eye. To minimize fatigue we gave subjects a short rest of approximately 30 seconds at the five minute mark.

6

Each subject visited the research facility three times. Each visit was spaced no less than two days and no more than three weeks apart. Table 1 outlines the tests performed at each visit.

Table 1. Tests that were performed at each of three visits.

Visits	Tests
1	physical and vision examinations screening fields out of chamber
2	preliminary fields in chamber dilated fundus examination
3	experimental fields in chamber

At the first visit, we explained to each applicant the test procedures and obtained a signed informed consent form. In order to eliminate potential subjects who may have had a physical or visual abnormality that would interfere with testing, the following procedures were performed at the first visit:

1. the physical examination;
2. vision examination including a subjective refraction, funduscopy through an undilated pupil, slit lamp examination of the anterior segment and tonometry;
3. visual field testing of both eyes (referred to as screening fields) with the central 24-2 threshold program.

At the conclusion of these procedures, the applicant was either dismissed or provisionally accepted into the study pending the outcome of the dilated fundus examination which was done at the second visit.

One eye of each subject was selected for further testing. In glaucoma subjects, we selected the eye that had the more clearly delineated depression unless the field loss was extensive in both hemifields in which case we selected the eye with the lesser field loss. One member of each pair of controls had their dominant eye tested while the other member had the non-dominant

eye tested. Half of the glaucoma subjects and half of the younger subjects had their dominant eye tested.

At the second visit, subjects underwent two successive visual field tests on the selected eye. These field tests were performed in the hypobaric chamber in order to habituate subjects to this environment. Atmospheric pressure remained at ground level (1290 feet) during these tests. Before each of the two preliminary field tests, subjects waited in the chamber for 24 minutes. This was done for all ground level tests to allow the same amount of pretest time and dark adaptation as would later be experienced during chamber decompression and equilibration.

After preliminary field testing, we dilated the pupils of both eyes of each subject with 1% tropicamide and performed the following:

1. binocular indirect ophthalmoscopy,
2. slit lamp inspection of the lens and optic nerve,
3. polaroid and 35mm stereoscopic photography of the optic nerve.

The third visit was devoted to visual field testing of the selected eye under two conditions: at ground level and at a simulated altitude of 10,000 feet. The test sequence was altered so that half of the subjects were tested first at ground level followed by testing at 10,000 feet (down-up). The other half of the subjects were tested first at 10,000 feet followed by ground level (up-down).

For subjects tested in the up-down sequence, the pretest ascent and equilibration took 24 minutes. The ascent rate was 2,000 feet per minute to 6,000 feet. This was followed by a descent to 2,000 feet at 2,000 feet per minute to check for possible sinus and middle ear blockage. Everything being normal, the chamber was depressurized at 2,000 feet per minute to a simulated altitude of 10,000 feet. Total ascent time, including the ear check, was approximately 9 minutes. An additional period of about 15 minutes was spent at 10,000 feet before the field test to allow for equilibration of PAO_2. After field testing, the pressure in the chamber was returned to ground level, this 'descent' taking about 5 minutes. An additional period of 19 minutes was spent resting, so that the total interval between tests was always 24 minutes.

In the down-up sequence, subjects first rested for 24 minutes and then were tested at ground level. Ascent and equilibration took 24 minutes. This was followed by a visual field test at 10,000 feet.

Subjects left the chamber after the final visual field test and went to an examination room where visual acuity, ophthalmoscopy, and tonometry were performed. The subjects were then dismissed.

Data were analyzed with a repeated measures design of multiple analysis of variance using SPSS/PC V2.0 software. The contrast used for analysis was the difference between the "up" and the "down" fields. The parameters used to describe the visual field results are defined below.

RESULTS

All points in the visual field were classified as belonging to one of three types depending on their position. The central point was classified as foveal, the 32 remaining innermost points were classified as paracentral; and the outer ring of 20 points at about 22 degrees eccentricity were peripheral.

Glaucoma.

Table 2a shows the means of the three types of points, under various test conditions, for both glaucoma and control subjects. For all types of points, glaucoma subjects showed lower mean sensitivities than did controls.

Table 2a. Mean sensitivity for three types of points in the visual field for six subjects with glaucoma (gl.) and for twelve age-matched controls (contr.) by whether the test was done at ground level or at a simulated altitude of 10,000 ft.

	Fovea		Paracentral		Peripheral	
Test Condition	gl.	contr.	gl.	contr.	gl.	contr.
Ground	32.8	36.4	18.7	29.1	17.4	27.8
10,000 ft.	33.7	36.8	19.4	29.1	18.2	27.9

Altitude did not affect the mean sensitivity for any type of point. This was shown by running a separate multiple analysis of variance (MANOVA) for each of the three outcome variables, using a repeated measures design (table 2b).

Table 2b. Results of MANOVAs, repeated measure design, for the within-subject effect of altitude (ground level or at 10,000 ft.) and the between-subject effect of glaucoma (with or without glaucoma) on the mean sensitivity of three parts of the visual field. Shown are the F values (1, 16 degrees of freedom) and probabilities (p) that differences observed were due to chance.

	Variable					
Factor	Fovea		Paracentral		Peripheral	
Glaucoma	10.28,	p=.006	18.79,	p=.001	17.88,	p=.001
Altitude	2.96,	p=.104	2.68,	p=.121	1.39,	p=.256
Interaction	0.33,	p=.574	2.10,	p=.176	1.12,	p=.306

Glaucoma is known to affect the upper and lower hemifields independently[30], so that glaucomatous damage accumulates in one hemifield in advance of the other. The field, therefore, becomes asymmetric across the horizontal meridian. Two measures of the difference between the upper and lower hemifields were devised: the mirror image index and the asymmetry index. The mirror image index was the sum of the absolute values of the differences between each point in the inferior field and its corresponding mirror-image point in the superior field. The asymmetry index was the absolute value of the difference between the mean threshold of superior points and the mean threshold of inferior points. If altitude increased glaucomatous depressions in the visual field, these indices would increase in value for subjects with glaucoma but not for controls.

Table 3a shows the means of these two measures by altitude. Both indices were approximately five times larger in subjects with glaucoma than in controls. However, there was no evidence that altitude affected these indices (table 3b).

10

Table 3a. Means of two indices (mirror and asym.) that measure the difference between the superior and inferior hemifields (see text) for six subjects with glaucoma (gl.) and for twelve age-matched controls (contr.) by whether the test was done at ground level or at a simulated altitude of 10,000 ft.

Test Condition	Mirror		Asym.	
	gl.	contr.	gl.	contr.
Ground	256.2	61.5	214.7	50.4
10,000 ft.	250.5	59.0	216.0	48.1

Table 3b. Results of MANOVAs, repeated measure design, for the within-subject effect of altitude (ground level or at 10,000 ft.) and the between-subject effect of glaucoma (with or without glaucoma) on two indices (mirror and asym.) that measure the difference between the superior and inferior hemifields (see text). Shown are the F values (1, 16 degrees of freedom) and probabilities (p) that differences observed were due to chance.

Factor	Variable			
	Mirror		Asym.	
Glaucoma	12.67,	p<.001	19.32,	p<.001
Altitude	0.33,	p=.575	<.001,	p=.950
Interaction	0.05,	p=.827	.05,	p=.818

Two measures which indicated how subjects responded to the testing procedure were the short-term fluctuation and the time to complete the test. Short-term fluctuation is an output of the Humphrey STATPAC. It measures the consistency of the subjects' responses during a single test session by thresholding 10 pre-selected points a second time. The means of the short-term fluctuation and time to complete the test are shown in table 4a. Subjects with glaucoma took more time to complete the visual field test and were more variable in their thresholds than controls. Results of the MANOVA failed to show any effect of altitude on these variables (table 4b).

11

Table 4a. Mean short-term fluctuation and mean time in minutes to complete testing for six subjects with glaucoma (gl.) and for twelve age-matched controls (contr.) by whether the test was done at ground level or at a simulated altitude of 10,000 ft.

Test Condition	Fluctuation		Time	
	gl.	contr.	gl.	contr.
Ground	1.7	1.3	11.1	9.2
10,000 ft.	1.9	1.4	10.9	9.5

Table 4b. Results of MANOVAs, repeated measure design, for the within-subject effect of altitude (ground level or at 10,000 ft.) and the between-subject effect of glaucoma (with or without glaucoma) on short-term fluctuation and mean time in minutes to complete testing. Shown are the F values (1, 16 degrees of freedom) and probabilities (p) that differences observed were due to chance.

	Variable			
Factor	Fluctuation		Time	
Glaucoma	4.49,	p=.050	7.63,	p=.014
Altitude	2.02,	p=.175	0.07,	p=.801
Interaction	0.02,	p=.895	1.32,	p=.267

Age study

The 12 normal older subjects, used as age-matched controls for the glaucoma study, were compared to six younger subjects to investigate any possible age-dependent effect of altitude on the visual field. Mean sensitivities of the three types of points in the visual field are shown in Table 5a for young and old subjects by condition of the experimental test (at ground level or at 10,000 ft.). The MANOVAs (table 5b) showed that older subjects had lower sensitivities than younger subjects for peripheral and paracentral points. Foveal thresholds were equal for the two groups. Altitude had no effect on these sensitivities.

Table 5a. Mean sensitivity for three types of points in the visual field for six subjects less than 36 years of age (young) and for twelve subjects over 45 years of age (older) by whether the test was done at ground level or at a simulated altitude of 10,000 ft.

	Fovea		Paracentral		Peripheral	
Test Condition	young	older	young	older	young	older
Ground	36.8	36.4	31.1	29.1	30.7	27.8
10,000 ft.	36.7	36.8	31.2	29.1	30.5	27.9

Table 5b. Results of MANOVAs, repeated measure design, for the within-subject effect of altitude (ground level or at 10,000 ft.) and the between-subject effect of age (<36 or >45 years old) on the mean sensitivity of three parts of the visual field. Shown are the F values (1, 16 degrees of freedom) and probabilities (p) that differences observed were due to chance.

	Variable					
Factor	Fovea		Paracentral		Peripheral	
Age	0.04,	p=.845	19.39,	p<.001	8.53,	p=.010
Altitude	0.13,	p=.727	0.06,	p=.811	0.08,	p=.780
Interaction	0.69,	p=.420	<.01,	p=.967	0.18,	p=.680

Table 6a shows the short-term fluctuation and the time to complete the visual fields test for both groups of subjects by test condition and by order of testing. Younger and older subjects took the same length of time to complete the test and showed equal short-term fluctuation in thresholds. Altitude failed to affect these variables (table 6b).

Table 6a. Mean short-term fluctuation and mean time in minutes to complete testing for six subjects less than 36 years of age (young) and for twelve subjects over 45 years of age (older) by whether the test was done at ground level or at a simulated altitude of 10,000 ft.

| | Fluctuation | | Time | |
Test Condition	young	older	young	older
Ground	1.1	1.3	8.9	9.2
10,000 ft.	1.0	1.4	8.8	9.5

Table 6b. Results of MANOVAs, repeated measure design, for the within-subject effect of altitude (ground level or at 10,000 ft.) and the between-subject effect of age (<36 or >45 years old) on the mean short-term fluctuation and mean time to complete testing. Shown are the F values (1, 16 degrees of freedom) and probabilities (p) that differences observed were due to chance.

| | Variable | | | |
Factor	Fluctuation		Time	
Age	5.94,	p=.027	1.75,	p=.205
Altitude	0.39,	p=.543	0.49,	p=.495
Interaction	1.83,	p=.191	0.88,	p=.362

DISCUSSION AND CONCLUSIONS

This study is concerned with the hypothesis that the relative hypoxia encountered in flight might temporarily increase the size of glaucomatous field defects, making glaucoma patients potentially unsafe as pilots. Also, if true, repeated flights may stress the optic nerve enough to accelerate glaucomatous field loss, a concern to passengers as well as pilots with glaucoma.

14

We selected 10,000 feet as our simulated test altitude because this would provide a margin of error in determining if visual field changes would be expected at altitude conditions more typical of commercial flight in pressurized aircraft (about 8,000 feet). General aviation pilots may fly to 12,500 feet without supplemental oxygen or pressurization but this altitude was not selected in this study to preclude unnecessary risk to the subjects. A simulated altitude of 10,000 feet did not have an effect on any of the eight visual field variables we measured.

Our results argue against any increased restrictions in the medical standards used by the FAA to medically certify civilian pilots with glaucoma. Although the Guide for Aviation Medical Examiners[31] does not automatically disqualify pilots from any class of license because of glaucoma, an ophthalmological consultation is required if the IOP is greater than or equal to 24 mm Hg or if there is a pressure difference greater than or equal to 5 mm Hg between the two eyes. If the ophthalmological report documents that pressures are under control and there is little or no field loss, the pilot may be certified. Our results showed that field loss revealed by testing at ground level did not increase with a brief exposure to an altitude greater than that experienced by most general aviation and commercial pilots. This was equally true both for older subjects with and without glaucoma and for younger normal subjects.

Current FAA medical certification policy does not appear to put pilots or passengers with glaucoma at risk for disease progression. Under short-term exposure to mild hypoxia, we could find no short-term change in visual field thresholds. Therefore we would not expect altitude to cause any long-term progression in glaucomatous damage.

REFERENCES

1. Siesjo BK, Johannsson H, Ljunggren B, Norberg K. Brain dysfunction in cerebral hypoxia and ischemia. In: Plum F, ed. Brain Dysfunction in Metabolic Disorders. New York: Raven Press. 1974: 74-112.

2. DeHart RL, ed. Fundamentals of Aerospace Medicine. Philadelphia: Lea & Febiger, 1985: 93.

3. McFarland RA. The effects of altitude on pilot performance. In: Hannisdahl B, Sem-Jacobsen CW, eds. Proceedings of XVII International Congress on Aviation and Space Medicine. Oslo: Universitetsforlaget, 1968: 96-108.

4. Ernsting J, Stewart WK. Hypoxia and altitude. In: Gillies GA, ed. A Textbook of Aviation Physiology. London: Pergamon Press, 1965: 209-13.

5. McFarland RA. Human factors in relation to the development of pressurized cabins. Aerospace Med 1971; 42:1303-18.

6. Ernest JT, Krill AE. The effect of hypoxia on visual function: psychophysical studies. Invest Ophthalmol 1971; 10:323-28.

7. Hecht S, Hendley CD, Frank SR, Haig C. Anoxia and brightness discrimination. J Gen Physiol 1946; 29:335-51.

8. Kohfeld DL. An evaluation of visual contrast sensitivity measures. In: Eberts RE, Eberts CG, eds. Trends in ergonomics/ human factors II. Amsterdam: Elsevier, 1985:83-92.

9. Kobrick JL, Crohn E, Shukitt B, Houston CS, Sutton JR, Cymerman A. Operation Everest II: lack of an effect of extreme altitude on visual contrast sensitivity. Aviat Space Environ Med 1988; 59:160-4.

10. McFarland RA, Halperin MH. The relation between foveal visual acuity and illumination under reduced oxygen tension. J Gen Physiol 1940; 23:613-30.

11. Evans JN, McFarland RA. The effects of oxygen deprivation on the central visual field. Am J Ophthalmol 1938; 21:968-80.

12. McFadden DM, Houston CS, Sutton JR, Powles ACP, Gray GW, Roberts RS. High-altitude retinopathy. JAMA 1981; 245:581-86.

13. Sutton JR, Coats G, Gray GW, Mansell AL, Powles ACP, Zahoruk R. Retinal studies at 446 torr in a hypobaric chamber. Aviat Space Environ Med 1980; 51:407-408.

14. Eysel UT. Susceptibility of the cat's visual system to hypoxia, hypotonia and circulatory arrest. Pflugers Arch 1978; 375:251-6.

15. Alder VA, Constable IJ. Effect of hypoxia on the maintained firing rate of retinal ganglion cells. Invest Ophthalmol Vis Sci 1981; 21:450-6.

16. Enroth-Cugell C, Goldstick TK, Linsenmeier RA. The contrast sensitivity of cat retinal ganglion cells at reduced oxygen tensions. J Physiol 1980; 304:59-81.

17. Noell WK. Site of asphyxial block in mammalian retinae. J Appl Physiol 1951; 3:489-500.

18. Linsenmeier RA, Mines AH, Steinberg RH. Effects of hypoxia and hypercapnia on the light peak and electroretinogram of the cat. Invest Ophthalmol 1983; 24:37-46.

19. Linsenmeier RA. Effects of light and darkness on oxygen distribution and consumption in the cat retina. J Gen Physiol 1986; 88:521-42.

20. Steinberg RH. Monitoring communications between photoreceptors and pigment epithelial cells: effects of "mild" systemic hypoxia. Invest Ophthalmol 1987; 28:1888-1904.

21. McFarland RA, Evans JN, Halperin MH. Ophthalmic aspects of acute oxygen deficiency. Arch Ophthalmol 1941; 26:886-913.

22. Merceir A, Perdriel G, Sole P, Chevaleraud J, Graveline J. Glaucoma incidence and significance in aviators. Aerospace Med 1964; 35:567-71.

23. Payne BF. Glaucoma as an aviation hazard. Aerospace Med 1962; 33:1328-31.

24. Dieckert JP, O'Connor PS, Schacklett DE, Tredici TJ, Lambert HM, Fanton JW, Sipperley JO, Rashid ER. Air travel and intraocular gas. Ophthalmology 1986; 93:642-5.

25. Hanscom TA, Diddie KR. Mountain travel and intraocular gas bubbles. Am J Ophthalmol 1987; 104:546.

26. Anderson DR. The mechanisms of damage of the optic nerve. In: Krieglstein GK, Leydhecker W, eds. Glaucoma Update II. New York: Springer-Verlag, 1983:89-93.

27. Nakagawara VB. The relevance of vision defects in the medical certification of civilian airmen. In: Goss DA, Edmondson LL. eds. Proceedings of the 1988 Northeastern State University Symposium on Theoretical and Clinical Optometry. Northeastern State University, Tahlequah, Ok. 1988:59-71.

28. Heijl A, Lindgren G, Olsson J. Normal variablility of static perimetric threshold values across the central visual field. Arch Ophthalmol 1987; 105:1544-9.

29. Balazsi GA, Rootman J, Drance SM, Schulzer M, Douglas GR. The effect of age on the nerve fiber population of the human optic nerve. Am J Ophthalmol 1984; 97:760-6.

30. Sommer A, Enger C, Witt K. Screening for glaucomatous field with automated threshold perimetry. Am J Cpthalomol 1987; 103:681-4.

31. Anonymous. Guide for Aviation Medical Examiners. US Dept of Transportation, Federal Aviation Administration. Oct. 1981:62-66.